

If Doctor Jellen had done no more than compile the classified list of pathological conditions to be found in the ileocecal region, he would have done us all a real service.

HENRY SNURE, M. D. (333 South McCadden Place, Los Angeles).—Lesions of the ileocecal region, as the author states, present a difficult problem in diagnosis even with the aid of a roentgenologic study, particularly when pain and palpable mass in the lower right quadrant are present. If the case is a chronic one, ample time for the complete roentgenologic study may be available and the correct diagnosis arrived at. Too often in hospital work the patient is admitted as an emergency with a request for a scout film of the abdomen to rule out obstruction and is in no physical condition for a prolonged roentgen study; many times a prompt exploratory operation is advisable rather than any further roentgenologic examination which may or may not provide a correct answer as to the type of pathology present.

Several years ago I made films of an infant, showing free air in the abdomen. Autopsy later showed a pin-point perforation of the cecum, the cause of which could not be determined. In this case operation was promptly done, but did not save the patient's life nor establish definitely the cause of perforation.

Doctor Jellen lists some forty types of lesions that may be present in the ileocecal region; fortunately some of these lesions occur rather infrequently, thereby reducing the percentage of possible errors in diagnosis. One point that should be emphasized is that the lymphoblastomas give no characteristic roentgenographic appearance. The diagnosis is usually made by biopsy at the time of operation.

I was glad to hear Doctor Jellen state that the diagnosis of chronic appendicitis is only occasionally offered by the roentgenologist; the various changes in the roentgenographic shadow of the appendix are of little value, location of pain point over the appendix and that shifts with the appendix is of value. He also states that the roentgenographic appearance of an appendiceal abscess is usually a smooth pressure defect on the mesial side of the cecum. However, I have occasionally found an irregular defect of the cecal outline laterally in appendiceal abscess that suggested carcinoma.

The author deserves credit for the very thorough and comprehensive manner in which he has presented the lesions of the ileocecal region.

## VITAMIN-DEFICIENCY STATES: THEIR RECOGNITION AND TREATMENT\*

By DWIGHT L. WILBUR, M.D.  
San Francisco

DISCUSSION by William C. Boeck, M. D., Los Angeles;  
Albert H. Rowe, M. D., San Francisco.

### PART II†

#### DEFICIENCY OF P-P FACTOR (VITAMIN G, B<sub>2</sub>, NICOTINIC ACID?)

##### Symptoms.

DEFICIENCY of this factor leads to the development of pellagra, the characteristic symptoms of which are three D's—diarrhea, dementia, and dermatitis—but these occur simultaneously in severe cases only. Perhaps the most characteristic feature is the dermatitis, without which the diagnosis is questionable. The cutaneous lesions consist of a dermatitis suggesting sunburn; a reddened, dirty brown skin, parchment-like, rough and scaling and affecting principally the exposed surface, especially the backs of the hands, wrists and forearms, usually in a symmetrical fashion. The face, neck, genitalia, and legs may be similarly affected. Gastro-intestinal symptoms which accompany pellagra are variable, although severe glossitis and

diarrhea are the most common. The stomatitis may consist of a brick-red color of the tongue, gums and cheeks, or there may be, in addition, considerable edema, ulceration, and exudation. Diarrhea may be intractable. Anorexia, vague abdominal distress and, at times, constipation may be present. Loss of weight is common, as are also nervous symptoms such as those of peripheral neuritis or of neurasthenia, namely, exhaustion, lassitude, and insomnia. The occurrence of any of these symptoms, accompanied by dermatitis of the type mentioned, should suggest the possibility of pellagra.

##### Diagnosis.

The diagnosis of pellagra depends entirely on the symptoms. While diagnostic and laboratory tests have not been developed, therapeutic test with nicotinic acid is proving of considerable diagnostic value.

Mild degrees of deficiency of the P-P factor are not clear-cut and little is known about them. The occurrence of otherwise unexplained glossitis, or vague gastro-intestinal symptoms with diarrhea, with or without symptoms referable to the nervous system, have been thought by some observers to be due to early pellagra or deficiency of vitamin G, (B<sub>2</sub>). The therapeutic effect of nicotinic acid in such cases may be of considerable diagnostic value if psychic influences can be controlled.

##### Treatment.

The daily requirement of the P-P factor or of nicotinic acid is unknown; nevertheless, the treatment of pellagra has become very effective, largely as a result of the work of Spies and his associates. It has been shown that the effective measures in treatment are a diet high in calories, proteins and vitamins (3,500 to 4,000 calories), supplemented by nicotinic acid in doses of 500 to 1,000 milligrams, by thiamin chlorid in doses of 20 to 50 milligrams, by powdered brewers' yeast in doses of 75 to 150 grams, or by dilute liver extract in doses of 25 to 75 cubic centimeters daily. Early in the course of the treatment the vitamin supplements may be administered parentally to those patients who, because of a sore mouth or vomiting, cannot tolerate an adequate diet. Bed rest, good nursing care and symptomatic treatment, consisting of sedatives, antiseptic solutions (such as potassium permanganate 1:5000 applied to the skin, and tincture of opium in large doses to control diarrhea), are also valuable adjuncts in treatment.

Since pellagra of the secondary type occurs not uncommonly in association with organic lesions in the gastro-intestinal tract, particularly with those which produce obstruction and with malignant disease of the stomach and colon, it is important to exclude gastro-intestinal disease before dismissing the patient.

##### Anemia—Abnormalities in the Gastro-Intestinal Tract.

Little is known of the etiologic factor or factors leading to the development of a macrocytic anemia, which is commonly observed in pellagra, beriberi, and other cases of deficiency of the vitamin B complex. The relation between this type of anemia and that of pernicious anemia is also not clear. How-

\* Read before the Pediatric Section of the California Medical Association at the sixty-seventh annual session, Pasadena, May 9-12, 1938.

† Part I appeared in the March, 1939, issue, page 184.

TABLE 7.—*Vitamin C Deficiency*

Degree of Deficiency	Clinical Signs	Incidence	Tests
Marked	Scurvy	Rare	1. Capillary resistance
Moderate	Infantile scurvy Hemorrhagic states Dental caries  Anemia  Increased susceptibility to infections	Unknown	2. Urinary excretion  3. Plasma levels 4. Intradermal 5. X-ray of bones 6. Therapeutic
Mild (Preclinical?)	Uncertain Above in milder form	Unknown	

ever, some factor in the vitamin B complex, but not thiamin, seems responsible for the macrocytic anemia of pellagra, beriberi and similar deficiency states.

Apparently gastro-intestinal symptoms, such as anorexia, glossitis, achlorhydria, hypomotility and hypotonicity, particularly of the small intestine, and diarrhea, are commonly associated with deficiency of the vitamin B complex. In some patients the clinical picture is characteristic of pellagra, in others it is not. Whether deficiencies of thiamin and of nicotinic acid will explain all of these symptoms is not clear.

#### VITAMIN C

##### *Symptoms.*

The most readily appreciated symptoms of deficiency of the antiscorbutic vitamin have to do with hemorrhagic phenomenon. This is due to the fact that the fundamental pathologic change in vitamin C deficiency is in the intercellular substance, particularly of the blood vessels, with softening of this substance which leads to hemorrhage into the subcutaneous, subperiosteal, gingival and other tissues. It has been observed that the intercellular substance, which ordinarily exists as a solid or gel, under circumstances of vitamin C deficiency may soften and become a liquid. Consequently, there has been much discussion of the possible rôle of vitamin C deficiency in acute and chronic hemorrhagic phenomena of the so-called "essential" or idiopathic type, and as to whether vitamin C deficiency may be a contributing factor in leading to hemorrhages in cases of peptic ulcer, menorrhagia, essential hematuria, subdural hematoma, and so forth. (Table 7.)

Vitamin C deficiency has also been connected with dental caries, anemia, and with increased susceptibility to infections. Most patients with infections require vitamin C in larger than usual quantities if normal levels are to be maintained in the blood and if normal quantities are to be excreted in the urine. Most observers are inclined to the opinion that this is due to the increased demand for vitamin C as a result of the infection, while others are inclined to the belief that a state of partial vitamin C deficiency may have predisposed to the development of the infection.

##### *Diagnosis.*

The clinical diagnosis of scurvy is relatively simple in a well-developed case. However, the clinical diagnosis of latent or preclinical scurvy is not so easy, although it may be suspected in patients who have unexplained hemorrhages of any type or in any situation, who have long-continued infections, or who have considerable dental caries. It is exceedingly important to emphasize that such conditions do not establish the diagnosis of vitamin C deficiency, but only suggest the possibility of it.

Fortunately, we possess better methods of determining the presence or absence of partial deficiency of vitamin C than of any other vitamin. These methods include the capillary resistance test, roentgen-ray studies of bones, intradermal test, studies of the levels of vitamin C in plasma and of the excretion of the vitamin in the urine, and vitamin C tolerance tests.

One simple method of performing the capillary resistance test in the office is to mark an area on the skin, 60 millimeters in diameter, in the antecubital fossa of one forearm. Following this, venous stasis is produced for fifteen minutes with a sphygmomanometer cuff at a pressure of 50 millimeters of mercury. If more than eight petechiae develop on the marked area the test is positive; if less than five develop, it is negative. Although this test is not specific, it is suggestive, and if positive in a case in which vitamin C deficiency is suspected, it should lead to studies either of vitamin C saturation or to treatment with vitamin C.

Methods of determining the vitamin C content of the plasma and the urine depend on the titration of these fluids with the indicator, 2-6 dichlorophenolindophenol. The titration of the urine is relatively simple and may be done in the physician's office if proper precautions are taken to preserve the urine in a dark bottle containing 10 per cent by volume of glacial acetic acid. The amount of vitamin C excreted in the urine by the normal individual in twenty-four hours is approximately 20 to 30 milligrams, while the vitamin C content of the blood plasma of the normal individual is in the neighborhood of 0.7 to 1.3 milligrams per hundred cubic centimeters of blood.

The most satisfactory method of determining vitamin C deficiency is in a measure of these levels and in an estimation of the tolerance of the patient to a test dose of vitamin C.

The diagnosis of very mild states of vitamin C deficiency depends entirely on such a chemical determination because clinical symptoms are lacking in these cases. There has been considerable discussion of the real clinical significance of such abnormal tests, for, as Finkle has pointed out, a fairly large proportion of the population suffers from an undersaturation of vitamin C. He believes that as yet there is no evidence to justify the conclusion that vitamin C deficiency has a causal relation to any pathologic condition other than scurvy.

##### *Treatment.*

Vitamin C deficiency may be avoided by intake of a diet adequate in fresh fruits, particularly of the citrus variety, and vegetables. The minimal intake necessary to prevent signs of deficiency is

TABLE 8.—Vitamin D Deficiency			
Degree of Deficiency	Clinical Signs	Incidence	Tests
Marked	Rickets Osteomalacia	Common Rare	1. X-ray of bones
Moderate	Rickets Dental caries Inadequate mineralization of bone Tetany	Common (50-90 per cent of infants)	2. Blood phosphatase 3. Therapeutic
Mild (Preclinical?)	Uncertain (Above in milder forms?)	Unknown	

not certain, but probably approximates 20 to 40 milligrams daily.

In the treatment of deficiency the diet should be high in its content of the above-noted foods, which may be supplemented by the administration of vitamin C in synthetic crystalline form, by mouth or intravenously, in doses of from 50 to 200 milligrams. In cases with hemorrhage in which rapid saturation of the tissues with vitamin C is desired, larger doses, such as 1000 milligrams, may be given daily for two to three days and then reduced to levels of from 100 to 300 milligrams daily until the therapeutic response is complete.

VITAMIN D DEFICIENCY

Symptoms.

Vitamin D is the antirachitic vitamin which plays an essential rôle in the metabolism of calcium and phosphorus, and has to do in particular with the retention of calcium and phosphorus in the body, in the deposit of these substances in bones and, perhaps, in the concentration of these elements in the blood. (Table 8.)

Rickets is such a widespread condition in infancy (50 to 90 per cent of infants are stated to have it) that it may be classified as probably the most common deficiency disease. Vitamin D deficiency in adults may lead to osteomalacia, which is very rare in the United States. It is probable also that certain forms of osteoporosis, of tetany, and possibly of dental caries, are the result of vitamin D deficiency, either because of an inadequate diet or because of interference with absorption of the vitamin or of calcium from the intestine.

Diagnosis.

The diagnosis of rickets is usually simple. When the classical symptoms are present, the diagnosis is obvious clinically. In cases in which the symptoms are less clear-cut, the diagnosis may be suspected clinically and confirmed by roentgenologic studies of the bones. Further confirmatory evidence may be obtained by determination of the blood phosphatase, which usually is increased in rickets. The therapeutic test with the administration of vitamin D may be helpful in establishing the diagnosis.

States of partial deficiency of vitamin D may be suspected if persons have dental caries, tetany, frequently occurring fractures or osteoporosis of the bones. During pregnancy or lactation, and among patients who are jaundiced or who have abnormalities of intestinal absorption such as occur in celiac disease and sprue, deficiency of vitamin D is most likely to be present. Whether the deficiency in such cases is the result of deficiency of calcium or phosphorus, or of abnormalities in function of the parathyroid glands may be difficult to determine. The administration of vitamin D may be helpful in distinguishing osteoporosis which is on a basis of deficiency from other types of osteoporosis, but a satisfactory response does not necessarily prove that the osteoporosis is attributable to vitamin D deficiency alone.

Treatment.

While there are at least ten different sterol derivatives which exhibit the properties of vitamin D, only two of these are known to be of prime importance in medicine. These are activated ergosterol or calciferol, which is the vitamin D of viosterol, irradiated yeast and yeast milk, and 7-dehydrocholesterol, which is the chief sterol of animal fats.

Rickets may be prevented by supplying the infant each day with one of the following: (1) two standard teaspoonfuls of cod-liver oil, which meets the requirements of the New and Nonofficial Remedies (1936); (2) one quart of milk containing 400 U. S. P. units of vitamin D to the quart; (3) five drops of viosterol (1.125 U. S. P. units); (4) irradiation of the skin with ultraviolet light.

In the treatment of rickets, a dose of twenty drops of viosterol (4500 U. S. P. units) or a similar number of units of vitamin D in another form is effective. Vitamin D may be supplied also in the

TABLE 9.—Vitamin Deficiencies				
Vitamin	Average Daily Requirement		Average Daily Therapeutic Dose	
	International units	Milligrams	International Units	Milligrams
A	4,000-10,000		25,000-75,000	
B <sub>1</sub>	200-500	1.0-2.0*	3,300-13,000	10-40
C	800-2,000	40-100	1,000-20,000	50-1,000
D	400-1,500 <sup>1</sup> 4,500 <sup>2</sup> Unknown <sup>3</sup>		3,000-4,000	
G (B <sub>2</sub> )			300-500 milligrams nicotinic acid 10-100 grams brewer's yeast 10-75 cubic centimeters dilute liver extract	

\* Mg. of synthetic HCl    <sup>1</sup> Average full-term infant    <sup>2</sup> Premature infant    <sup>3</sup> Child and adult

TABLE 10.—*Protective Foods Which Serve as a Foundation for a Normal Diet (Daily Quantities)*

Milk	1 pint
Egg	1
Vegetables	3 large servings, besides potatoes (one a green leafy vegetable)
Fruit	2 servings (one raw)
Meat, fish or fowl	1 serving (about 2 ounces)
Butter	1 tablespoonful

form of fish-liver oils, or irradiated yeast, milk and cereals, of milk to which a concentrate of vitamin D has been added or by irradiation of the skin.

In adults with senile osteoporosis, ten drops of viosterol three times daily in conjunction with four drams of calcium lactate or tribasic calcium phosphate three times daily often will relieve symptoms.

#### DEFICIENCIES OF OTHER VITAMINS: VITAMIN E

At least three substances possess the effect of vitamin E, but as yet it has not been clearly demonstrated that any of them is required by man for normal health or in reproduction. Of principal clinical interest is the possible etiologic rôle of deficiency of vitamin E in threatened and spontaneous abortion, and of an excess of the vitamin in malignant disease. It is difficult to obtain proof that vitamin E is of value in the treatment of sterility and habitual abortion in human beings, and much more evidence is greatly needed to establish the usefulness of vitamin E in abnormal human reproduction.

#### VITAMIN K

Apparently the antihemorrhagic factor, known as vitamin K, may play some rôle in the production of prothrombin, for in certain species of animals deficiency of vitamin K leads to a decrease in the prothrombin content of the blood. In considering the possible therapeutic use of vitamin K in patients with jaundice it has been suggested that it is of value because the prothrombin deficiency of such patients may be due to deficiency of vitamin K, since vitamin K is fat-soluble and there is frequently interference of absorption of fat in patients with jaundice. Vitamin K is not available on the market, but substances rich in it are green vegetables, pig-liver fat, hemp seed, and alfalfa.

#### USE OF VITAMINS

##### *Use of Vitamins in Persons Who Are Chronically Fatigued, Rundown, Underweight, Below Par or Subject to Frequent Infections.*

The diagnostic and therapeutic problems presented by patients who fall into this group are often very difficult to solve. It goes without saying that great care must be used to exclude the presence of an underlying organic disease when these symptoms are present, and adequately to treat it. It is probable that, of the patients who fall into this group, a certain number will present no distinguishable organic disease. Some of this latter group of patients may be benefited by administration of vitamins while other patients may not, depending on whether or not in a particular case the symptoms

are due to vitamin deficiency and depending to some extent on psychic influences.

How is one to tell which patients will do well and which poorly on vitamin therapy? There is no clear-cut way to distinguish between these groups at the present time. However, the history of an inadequate diet, the presence of symptoms or findings of mild deficiency states or the occurrence of abnormal responses to some of the diagnostic tests previously mentioned may be helpful in determining the patients who may benefit from vitamin therapy.

It is important to recall that the conditions we discussing are symptoms rather than diseases, and that it is essential to treat any underlying cause of them. It is no more sensible to give vitamin therapy to all such patients and to expect good results than it is to give aspirin to all patients with headache, codein to all who cough, or opium to all who have diarrhea.

#### COMMENT

Finally, too much stress cannot be placed on the importance of an adequate diet in the prevention and treatment of deficiency diseases. It has been well said that the grocery store is more important than the drug store in the prevention and treatment of these conditions. In the treatment of some deficiency states, crystals and concentrates of some of the vitamins are available and, while these products are extremely useful, they should always be supplemented by vitamins in the natural state, *i. e.*, in foods, yeast, cod-liver oil, and so forth, not only because this is the way in which vitamins ordinarily are obtained by normal persons, but especially because there are present in foods certain essential substances which have not only not been synthesized or concentrated, but of which at present we are only vaguely aware. (Tables 9 and 10.)

490 Post Street.

#### DISCUSSION

WILLIAM C. BOECK, M. D. (2210 West Third Street, Los Angeles).—I agree heartily with the substance of Doctor Wilbur's excellent paper on the very timely subject of vitamin-deficiency states.

I have been led to look upon vitamin-deficiency states of two kinds, namely, (1) clinical and (2) subclinical or "pre-clinical," depending upon the degree of deficiency that exists.

Clinical vitamin-deficiency states comprise the well-known group of avitaminoses with their classical, characteristic symptoms, signs and pathology. In this group we recognize such avitaminoses as xerophthalmia and hemerolopia (vitamin A deficiency); beriberi (vitamin B<sub>1</sub>); scurvy (vitamin C); rickets (vitamin D), etc.

The suspicion that these conditions might exist in a given patient comes from a good history of an inadequate intake or absorption of food rich in the specific vitamins. These deficiency states, the clinical avitaminoses, are not difficult to diagnose, since each has its own characteristic syndrome and pathology, but there is a feeling that vitamin-deficiency states of mild degree exists, which are often not clear syndromes of symptoms and signs. These comprise the "sub-clinical," or, as Doctor Wilbur calls them, "preclinical" vitamin-deficiency states.

We read of the protective action of vitamins A and C against infections in general, in pneumonia, myasthenia, and treatment of rheumatoid arthritis, to immunity processes, hemorrhage, purpura, anemia, dental caries, etc.; the relation of vitamin B<sub>1</sub> to such conditions as anorexia, cardiac irregularities, atonic constipation, diarrhea, edema, low-serum proteins, fatigue and asthenia, anemia, lactation, growth, etc.; the relation of vitamin D to nervous irritability, asthenia, to tubercular resistance and other infections, calculi, caries, etc.; and the relation of vitamin E to steril-

ity, impotence, miscarriage, asthenia, loss of vigor and weight, muscular atrophy, etc. One could go on almost indefinitely enumerating many other conditions which have been attributed to vitamin deficiency, until one might be almost convinced that all body ills are due to vitamin deficiency. Since the major portion of our population possesses some of the disturbances mentioned as characterizing some degree of vitamin deficiency, it is not surprising to learn that vitamin products rate fourth in value among drug-store sales, and that we appear to be in an era of a new fad—that of vitamins.

Obviously, the status of vitamins in this preclinical group of deficiency states has led to considerable confusion because of lack of (1) correct diagnosis, very often of the underlying cause, since the clinical improvement or cure which has been often erroneously attributed to the vitamin therapy, in these cases, may actually be due to some other corrected condition; (2) lack of uniform treatment of these conditions with vitamin-containing products has led to further confusion because the vitamin therapy has been variable as to method of administration, source of the vitamins used, and dosage employed in different standardized units.

That considerable confusion results from an incorrect diagnosis of the underlying functional or pathological disturbance is apparent at once. All cases of hemerolopia, or night blindness, are not due to vitamin A deficiency, for, as Frandsen and later Yudkin have pointed out, it occurs in persons with neurasthenia and hysteria, in poisoning from quinin, alcohol, nicotine, in retinitis and choroiditis, retinitis pigmentosa, glaucoma, excessive myopia, and many other conditions. Therefore, in considering treatment of persons showing hemerolopia by the biophotometer, the underlying cause must be determined.

It will do no harm to give vitamin A, since it is a food factor; but if improvement is noted it may not always be due to dietary treatment given. The ophthalmologist should also be called in to treat any underlying pathology or disturbance in visual acuity.

Again vitamin B<sub>1</sub> deficiency, as Cowgill states, does not explain all cases of anorexia, atonic constipation, edema, cardiac enlargement, fatigue, anemia, etc. To administer it in all such cases does no bodily harm, but it does not answer the question as to the etiology of the symptoms. One could go on with instances regarding the other vitamins, C and D, and B<sub>2</sub> as well, and come to realize that a correct diagnosis is the primary essential to an understanding of these cases before we can conclude they are always due to a preclinical state of vitamin deficiency.

We are handicapped in recognizing the mild states of vitamin deficiency because the clinical tests for vitamins A, B, C, and D are not available to everyone, and are based mostly on animal-growth observations, which admit of much variability at their best. There are, as yet, no well-recognized standards of value for the presence and excretion of these vitamins in normal human beings to serve as a dependable guide or basis for comparability. All this needs more investigation.

The treatment of vitamin-deficiency states, both clinical and preclinical, has caused more confusion: first, as to method of administration, by mouth as part of the diet rich in the particular vitamin, or parenterally by use of the purified product. For example, from 10 to 100 milligrams daily of thiamin chlorid have been used to cure polyneuritis of B<sub>1</sub> deficiency, but no one has shown how much is necessary; and large doses, while effective, may be too expensive at present for many patients.

The confusion has been added to by the fact that all vitamin products on the market, while purporting to represent so many units per gram, or cubic centimeter or tablet, are often deficient when subjected to animal-assaying procedures of standardization. This may be due to factors that are uncontrollable because animals are used, and because the United States Pharmacopeia reference standards may not yield all the vitamins they are supposed to yield. The purified vitamins will, doubtless, prove to be better reference standards in the future, but Williams feels that in the case of vitamin B<sub>1</sub> it will be difficult to find a satisfactory method of chemical assay, since it possesses no known physical property which is adopted to delicate testing. Better United States Pharmacopeia reference standards to determine vitamin units would also do away with

the confusion still existing in reference to the labeling of these vitamin concentrates in different kinds of units; this practice, however, is becoming less prevalent.

May I summarize by stating: (1) That the clinical types of vitamin deficiency, the classical avitaminoses, are not difficult of diagnosis and treatment, because of their characteristic symptoms, signs, and pathology, although the matter of correct dosage is still obscure; and that (2) the "pre-clinical" types are difficult of diagnosis, because the symptoms are also those of other functional or pathological disturbances, and hence vitamin concentrates may be given, but often unnecessarily. This leads to confusion, but produces no bodily harm, although the patient may suffer an unnecessary financial relapse since vitamin products are expensive. (3) It is hoped that the future will provide better methods for the detection of these preclinical vitamin-deficiency states, since the present methods of detection are not available to all, and are not based upon accurate standards. Then experimental and therapeutic procedures will be far more comparable, and therefore more valuable, because of uniformity of the source, dosage, and administration of the vitamin products. (4) Let us all keep in mind the substance of Doctor Wilbur's Tables 2 and 3, which afford a most excellent guide to the question of when we may suspect a state of vitamin deficiency, because if this guide is followed much unnecessary vitamin administrations will be avoided.

✽

ALBERT H. ROWE, M. D. (490 Post Street, San Francisco).—The various manifestations and possible results of a deficient vitamin intake, so well discussed by Doctor Wilbur in his article, necessarily should be in the minds of all physicians in the study and treatment of patients, for avitaminosis is possible in any individual. Doctor Wilbur's emphasis of the mild or subclinical results of deficient vitamins is especially timely. The fact that absorption of vitamins naturally occurring in food, and of those administered orally, may not be adequate for metabolic requirements, must be appreciated. Because of this, the increasing accuracy of clinical and laboratory tests, discussed by Doctor Wilbur, which reveal probable deficiencies in the important known vitamins in the body, offers valuable information which makes possible proper dietary adjustments and indicated supplemental vitamin therapy. In the endeavor to protect all patients against vitamin and other dietary deficiencies, every history should contain information concerning the past and present habits of diet. In such dietary history, the average amounts of foods of all kinds and types taken during previous decades, and during the past few years, allow a rough, though clinically valuable estimation of the intake of necessary vitamins throughout life, and the likelihood of protein or mineral deficiencies which may have contributed to chronic or present symptomatology. Such a diet history, moreover, should record detailed information concerning possible food dislikes or idiosyncrasies which suggest possible mild or more severe food allergies.

Of great help in the determination of the vitamin content of a diet is the tabulated number of units of vitamins A, B<sub>1</sub>, B<sub>2</sub>, C, and D in 100-gram amounts of most foods assembled by the United States Department of Agriculture in 1937, as reported in Bulletin A-275. The units of the various vitamins have been determined by many investigations and experimental studies, and offer approximate though practical information. With the daily average requirements of the vitamins known, as listed by Doctor Wilbur and as published elsewhere, it is usually possible to arrange the diet so that proper amounts of ordinary foods can satisfy the vitamin demand of the body. Frequently, additional vitamin therapy, especially with vitamin D, is required, especially if long-existing deficiencies have been present. Such assurance of adequate vitamins in the diet, as well as of minerals and proteins, is especially necessary when the diet must be modified because of metabolic, allergic, gastro-intestinal, or other diseases. This is particularly important in the use of elimination or test negative diets for the study or control of food allergy, in which absolute assurance of adequate vitamins, proteins, minerals, and calories to satisfy the patient's nutritional requirements is mandatory.